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-USSR-

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FOREWORD

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DETERMINATION OF THE PARTIAL PRESSURE OF GASES IN ARTERIAL BLOOD IN CERTAIN LUNG DISEASES

[Following is a translation of an article by E. Grants in Izvestiya Akademii nauk Latviyskoy SSR (Bulletin of the Academy of Sciences, Latvian SSR), No. 11 (148), 1959, Riga, pages 169-177.]

In studying the external respiratory function in the thoracic clinic greater importance is being given to examination of arterial blood. While the Knipping-Brauer school, or the so-called German school, with its successors (Ulenbruk, Vorwerk, Valentin, Wenrath, Haubatz, and others) perfected the spirographic methods for studying pulmonary and cardiovascular functions in various pathological states, some shortcomings of these methods were detected in the 1930's and mentioned several times in the literature. For instance, in the opinion of Rossier [27], the principal defect is the incorrect evaluation of the so-called spirographic deficit of oxygen which in many cases does not reflect the actual condition either quantitatively or qualitatively.

Recently, in order to get the most accurate data on the gas exchange and oxygen balance of the body, greater attention has been given to methods for determining the partial pressure of the respiratory gases in arterial blood.

The partial pressure of oxygen in arterial blood (pO_2 art.) is the value which best describes the state of the body's oxygen supply [18]. By determining the so-called alveolo-arterial difference (AaD), we can get an idea of oxygen diffusion in the lungs in the broad sense, and the degree of its impairment.

In determining the above-mentioned values, some methodological difficulties arose which have been largely, though not completely, overcome during the past 10-15 years. Quite some time ago, authors were already pointing out the inadmissibility of the use of arterial puncture for diagnostic purposes, referring to the possible complications [2, 12, 31], but it has been widely employed in practice. But for determining the oxygen content of arterial blood there has also been frequent use of the method of taking "arterialized" capillary blood from the finger or vein of a previously warmed hand [2, 21]. This method is of course not suitable for determining the partial pressure of gases.

If an artery is punctured under local anesthesia, we overcome the objections that the change in respiration during the puncture effects the gas content of the blood [12]. Much greater difficulties developed when we attempted to work out a rather simple method for determining the partial pressure of gases in the blood.

For a long time the pO_2 was determined from the oxyhemoglobin dissociation curve. We cannot deny that with an accurate determination of oxygen saturation without major disturbances of the acid-base balance and temperature it is possible to obtain an approximate value for pO_2 art. However, in the flat portion of the dissociation curve the results obtained are too inaccurate [28]. Keeping in mind the variations in the curve, we see that its determination is a complicated procedure in each instance.

For this reason attempts were renewed to use direct methods for determining partial pressure in whole blood. At present the microtonometric and potentiometric methods are widely used in practice. The potentiometric determination of pO_2 art., using Bartels' "hemooxytensimeter," makes it possible, according to the author's data [7, 8, 10], to determine pO_2 art. within a range of from 10 to 500 mm $\pm$ 2% within 3 minutes. However, this method has defects consisting in the necessity for determining the standard curve for each blood sample; in addition, this method cannot be used to determine pCO_2 art.

The pO_2 and pCO_2 art. for a single blood sample can be determined using the method developed by Riley and his colleagues [25] and subsequently somewhat improved [15, 24]. It is based on the principle employed by A. Krog as early as 1908: the equalization of the pressure of alveolar gases with the pressure of gases in a small amount of blood. In Riley's method the gas pressure of a small amount (1 ml) of blood is equalized by a bubble of alveolar air (7-10 mm^3), and then the bubble is analyzed for CO_2 and O_2. The method is quite accurate ($\pm$ 2.0 mm Hg) within a range of up to 160 mm Hg [8]. Inasmuch as we have not found this method described in Soviet literature, we believe it practical to spend a little more time in describing it in detail.

In the procedure the analyzers are employed which were proposed by Scholander and Roughton [30] for determining the O_2 and CO_2 content of the blood and which consist of a glass syringe (1-2 ml) with attached microcapillary tubes. It is desirable to have an analyzer capillary tube 0.5 mm in diameter, since broader capillary tubes may affect the accuracy of the results. Starting from the fact that the gas bubble added to the blood must not alter the partial pressure of gases in the apparatus, it was established [25] that the ratio between the bubble and the blood must be 1:141. If we take 1 ml of blood for analysis, then the volume of the bubble must not exceed 7-8 mm^3 in cases where the expected pO_2 falls within the range of 60-100 mm Hg and over. According to the authors of the method, one division on the capillary tube corresponds to 0.4 mm^3 and the length of the added bubble is 18-25 units. If a smaller bubble is used, the possibility of error increases. Later, a magnifying glass mounted in a millimeter scale was used for making the count [24].

In our work we used a capillary tube 0.7 mm in diameter. In order to maintain the accuracy of the method, we increased the volume of the gas bubble to 11-12 mm^3 per 1.5 ml of blood with a syringe capacity of 2 ml, and in counting used only a magnifying glass and a millimeter scale and not the divisions of a capillary tube. The blood was drawn from the ulnar artery by puncture under local anesthesia with a 1% novocaine solution; oxyhemometry was employed simultaneously. For an anticoagulant we used heparin with an admixture of sodium fluoride. The analyzers were filled with blood under anaerobic conditions immediately after the blood was drawn by means of a needle passed through a conical piece of rubber adhering tightly to the walls of the analyzer vessel. The "dead space" in the analyzer was in this instance filled not with mercury but with arterial blood.

For the analysis the alveolar air was collected in a rubber bag and introduced into the analyzer by means of the same needle. The gas bubble of 11-12 mm^3 volume was covered from above with blood and drawn into the syringe, after which the analyzer was placed in a water bath (+ 37.5 $\pm$ 0.2° C) and secured in a rotating device. This procedure lasted no more than 5-10 minutes. The blood with the bubble was rotated in the water bath for 5-7 minutes, after which almost all the blood was withdrawn from the analyzer into a separate vessel under water. By maintaining the vertical position of the analyzer, the gas bubble quickly passed into the capillary tube. The count was made under water. After measuring the volume of the bubble (V_1), the analyzer vessel was filled with distilled water, a part of which was drawn into the syringe. The vessel was filled with a 4% solution of NaOH to absorb the CO_2. This was done by repeatedly drawing the alkali into the capillary tube. After this, the capillary tube was again placed in the water bath for a second count (V_2). Thus by drawing the sodium hyposulfite solution (0.5 $Na_2S_2O_4$ dissolved in 2.5 ml of a solution of 5.6% KOH) the O_2 was also absorbed, after which the analyzer was again submerged in the water bath and the volume of the bubble measured a third time (V_3).

Partial pressures were computed on the basis of the following formulas:

$$\% CO_2 = \frac{V_1 - V_2}{V_1}; \; pCO \text{ mm Hg} = \% CO_2 \text{ (Ba-47)}$$

$$\% O_2 = \frac{V_2 - V_3}{V_1}; \; pO_2 \text{ mm Hg} = \% O_2 \text{ (Ba-47)}$$

A comparison of the data obtained with the tonometric data revealed the necessity for correcting the results by the empiric factors +2 mm Hg for CO_2 and -3 mm Hg for O_2. In our work we used a nomogram, which gives more accurate corrections [24].

It is recommended that two parallel analyses be made, and in a case where the degree of difference in the results exceeds the method error that a third analysis be made [29].

In obtaining the alveoloarterial difference (AaD) for oxygen, some difficulties arose in connection with determining the partial pressure of gases in alveolar air. Accuracy in determining these values frequently has a decisive importance in computing the AaD [10, 20, 17]. The long-used methodology developed by Holden and Priestley has frequently been subject to criticism [14, 15, 19] in connection with the nonuniform gas content of different portions of the alveolar air, as well as the decrease in a certain amount of O_2 during prolonged expiration.

In addition to the new methods for obtaining alveolar air [1, 6, 19, 22], Bartels has proposed a modification of the Holden-Priestley method [10, 11]. In addition, he proposed formulas for computing the gas composition of alveolar air by an indirect method. This computation is based on Benzinger's formula [28]. In our work we used the formula worked out by Riley and his colleagues [23]. The data thus obtained, in the authors' opinion, reflects the "effective" partial pressure of oxygen and carbon dioxide in the alveoli, and indicates the amount of pressures at which gas exchange is possible between the functioning alveoli and the blood of the pulmonary capillaries during several ventilation cycles:

$$p_{O_2}\text{ alv. effect.} = p_{O_2}\text{ trach.}\ \frac{\%\ N_2\text{ exp.}}{\%\ N_2\text{ insp.}} - \frac{p_{CO_2}\text{ art.}}{RE}$$

where p_{O_2} trach. is the p_{O_2} in inspired air (at a given atmospheric pressure, body temperature, total saturation with water vapor);

$\%N_2$ insp. is the percentage of nitrogen in inspired air;
$\%N_2$ exp. is the percentage of nitrogen is expired air;
p_{CO_2} art. is the partial CO_2 pressure in arterial blood;

We obtained data on the partial pressure of gases in alveolar air by parallel determination by the Holden-Priestley method with Bartels' modification and by Riley's formula (see Table 1). On the average, the computed and effective partial pressures for 25 patients with pulmonary diseases were 2.5 mm Hg higher than those which we obtained by the Holden-Priestley method. It should be pointed out that for the authors of the indirect method the p_{O_2} alv. effect. was higher (between 0-17 mm Hg), but in our cases (Nos. 4, 5, 8, 9, 21, and 23) it is clear that before exhaling into the apparatus the patient either inhaled briefly, which may explain the drop in CO_2 and the increase in O_2, or did not exhale deeply enough.

TABLE 1

Test No.	pCO_2 alv.	pO_2 alv.	pO_2 effect.	Difference
1	43.6	93.7	95.7	+ 2.0
2	46.0	88.4	98.6	+ 10.2
3	41.4	94.2	101.6	+ 7.4
4	40.6	105.1	87.6	- 17.5
5	40.0	91.0	84.8	- 6.2
6	43.6	78.4	96.6	+ 18.2
7	42.1	88.6	88.7	+ 0.1
8	38.7	105.6	93.6	- 12.0
9	36.6	101.0	96.0	- 5.0
10	40.7	90.3	100.8	+ 10.1
11	40.7	94.0	100.5	+ 6.5
12	41.5	83.8	95.8	+ 12.0
13	46.4	91.5	87.4	- 4.1
14	40.4	96.3	96	- 0.7
15	39.7	99.0	108.4	+ 9.4
16	35.8	95.2	98.4	+ 3.2
17	43.6	78.4	96.3	+ 17.9
18	40.70	100.1	104.6	+ 4.5
19	47.2	79.3	98.4	+ 18.7
20	42.6	99.0	102.1	+ 3.1
21	36.5	107.5	98.0	- 9.5
22	33.3	79.4	85.1	+ 5.7
23	40.1	101.8	88.0	- 13.8
24	48.5	76.7	84.8	+ 8.1
25	44.6	86.3	86.0	- 0.3

Average + 2.7

In order to avoid such cases in computing the AaD, we used only the value for the pO_2 alv. effect. when the source of error might in practice be the inaccurate determination of the PCO_2 art., but the inaccuracy rarely exceeds ± 3 mm Hg [23] for the computed pO_2 alv.

In determining the partial pressure of gases in arterial blood for 30 pulmonary patients we examined ventilation, gas exchange, the gas composition of the arterial blood, and oxhemometry. The principal data are cited in Tables 2-4.

As for the average figures for pO_2 art. and AaD under normal conditions there is something of a disparity in the data from the literature. Lilienthal and Riley and colleagues [17] believe that the pO_2 art. under normal conditions averages 94.2 mm Hg (83-102 mm Hg) and the AaD 9.3 mm Hg. Their data coincide with the data of Bartels and his colleagues [10, 11] whose obtained pO_2 art. averages 92, 95 (± 5.6) mm Hg and AaD 4.85 (± 5.41) mm Hg, and with that of Filley and his colleagues [14] whose obtained pO_2 art. equals 85.1 and AaD 9.7 mm Hg (in a range from 0 to 20). Rees and Black obtained a pO_2 art. fluctuating between 83.6 and 102.6 mm Hg [26].

In our work we considered the pO_2 art. reduced if it was below 80 mm Hg, which corresponds to indications in the literature [20], and the AaD increased if it exceeded 15 mm Hg.

We obtained our data on ventilation and gas exchange by the Douglas-Holden method. The indicated volumes of gases (CO_2 and O_2) were reduced to normal conditions (0°, 760 mm Hg, dry). Oxyhemometry was carried out by using the Soviet O-36 oxyhemograph: breathing 100% oxygen, the patient breathed from a sack through a respirator mouthpiece. Exercise consisted of going rapidly up and down three stairs for 5 minutes. In oxyhemometry we did not use the absolute figures of the scale but set the needle in an arbitrary position at the top of the scale, and in evaluating the results we considered only the relative rise and fall in the oxyhemoglobin content in order to exclude the possible errors which are mentioned in the literature [4, 13]. The appropriate tables give the maximum drop in the oxyhemoglobin content during exercise or during the following 2 minutes. In the graph, "oxygen breathing" indicates the time during which saturation reached its maximum, which to a certain degree makes it possible to judge the uniformity and effectiveness of the ventilation.

Using the value of pO_2 art. in evaluating the condition of the patient, we wished to obtain data on the degree of hypoxemia, if there was any, that would be more accurate than the existing data on the oxygen saturation of arterial blood.

Taking into account that when there is a change in the pH and pCO_2 art. there is also a change in the oxyhemoglobin dissociation curve, as known from the literature [3] (and for this reason the percentage of saturation does not always reflect sufficiently accurately the condition of the body's oxygen supply), we must consider the pO_2 art. obtained by the direct method to be the more accurate value.

In the literature we find proposals, deriving from the value of the pO_2 art., to divide hypoxemia into three degrees [18]:

1st degree hypoxemia--pO_2 art. 85-75 mm Hg
2nd degree hypoxemia--pO_2 art. 75-60 mm Hg
3rd degree hypoxemia--pO_2 art. below 60 mm Hg.

We also find recommendations to use this division in solving a number of problems of practical importance (operability, patient's conditions in the postoperative period, etc.). It should be pointed out that in the case of the author who proposed this division [18] all patients with surgical diseases of the lungs had more or less pronounced hypoxemia.

In judging the value of AaD, we believed that, along with other data from the examination, it may give an idea of the mechanism of the origin of hypoxemia. Bartels and others [11, 14, 20] came to the conclusion that the cause of the origin of a normal AaD, excluding the possibility of errors in methodology, is the admixture of nonarterialized blood in the arterial (for example, arteriovenous anastomoses, conservation of circulation in the poorly ventilaged portions of the lungs, etc.), as well as inhibited diffusion through the alveolar membrane (for example, an increase in the rate of blood flow in the pulmonary capillaries).

Apparently, the same factors cause the increase in the AaD in pathological states. While the presence of many pathological symptoms disrupting the function of external respiration can be determined by a number of spirographic and gas analyses, the greatest difficulties are encountered in attempting to determine the role of the "short circuit" factor in the occurrence of hypoxemia. The literature contains data which affirm that a shunt causes hypoxemia in a number of surgical diseases of the lungs [5].

In 1948 Rossier [27] demonstrated the possibility of detecting a shunt by using the respiration of pure oxygen. In cases where the alveoli, though insufficiently ventilated, have a connection with the outer air, the saturation in a rather prolonged period (10 minutes) sometimes reaches 100%.

In case of a shunt, the oxygen saturation may increase but never reaches 100%. It is impossible in this way to differentiate between a vascular and a cardiac shunt. In our material, signs of a shunt were observed principally in fresh infiltrates and atelectasis where circulation was still preserved. Most frequently, however, cases were encountered where there was a combination of a shunt with other factors which inhibit normal gas exchange [27, 28].

TABLE 2

No.	Patient	Diagnosis	Minute volume in ventilation ml	Oxygen absorption in ml	Carbon dioxide secretion in ml	Oxygen utilization coefficient	Percentage of saturation of art. blood	pCO_2 alv.	pO_2 alv.	pO_2 art.	AaD	Oxyhemometry: Breathing 100% oxygen	Oxyhemometry: during exercise
1.	K. K.	Pneumosclerosis of lower lobes of both lungs	6926	246	275	35.3	89.8	46.4	107	78	29	+4% in 4 min	-4%
2.	A. A.	Chronic pneumonia of right lung	5300	276	194	52.0	89.6	52.0	102	73	29	+2% in 2 min	-1%
3.	L. N.	Cancer of right lung with atelectasis of lower lobe	7430	299	242	40.1	90.2	40.2	88	68	20	+8% in 2 min	+1%
4.	Ya. L.	Multiple abscesses of the right lung	8900	256	237	26.6	82.4	44.6	87	56	31	+5% in 10 min	-4%
5.	K. V.	Bronchoectasis	9160	250	282	27.2	82.4	48.0	84	65	19	+4% in 2 min	-4%
6.	V. T.	Condition after right lower lobectomy	9300	352	266	37.8	87.5	41.6	86	71	15	+11% in 9 min	-2%
7.	A. B.	Abscessing pneumonia of left lung	8230	273	234	29.4	90.8	41.5	95	75	20	+9% in 4 min	-3%
8.	A. R.	3rd-4th degree silicosis Abscess of right lung	9560	302	257	31.5	88.1	37.5	97	70	27	—	—
9.	Ya. Z.	Abscess of right lung	8100	287	214	35.4	87.0	52.4	75	67	8	+10% in 3 min	+2%
10.	A. K.	Brochoectasis of lobes of both lungs	5900	162	156	28.9	93.8	39.7	108	95	13	+6% in 7 min	+3%

Regarding the 10 patients we examined who were suffering from chronic suppurative diseases of the lungs (see Table 2) we detected a drop in the pO_2 art., except in one: patient A. K. — even below the "critical value" of 60 mm Hg. The lowest figures (patients K. K., A. A., Ya. L., K. V., and A. R.) occurred when radiography and clinical observation revealed more or less pronounced inflammatory lesions (increase in temperature, leucocytosis, erythrocyte sedimentation reaction, coughing with excessive sputum, etc.). Even the data from the spirographic examination were lower, particularly the respiratory reserves. The clinical picture thus completely corresponded to the severity of hypoemia, to judge by the pO_2 art. In oxygen breathing it was found that in these cases the saturation rose by only 2-5%. Indications of a shunt occurred principally in patients with a fresh inflammatory process in the lungs or an exacerbation of a chronic one, which make it possible to assume that circulation was maintained in the affected area.

TABLE 3

No.	Patient	Diagnosis	Min. volume ventilation in ml	Oxygen absorption in ml	Carbon dioxide secretion in ml	Oxygen utilization coefficient	Percentage of saturation of art. blood	pCO_2 alv.	pO_2 alv. effect.	pO_2 art.	AaD	Oxyhemometry: Breathing 100% Oxygen	Oxyhemometry: During Exercise
1.	F. S.	Cancer of left lung (peripheral form).	5300	235	174	44.3	97.9	40.4	93	78	15	+3% in 2 min	-4% to +2%
2.	G. N.	Cancer of left lung (central form).	9630	297	224	30.9	93.2	44.0	86	79	7	+6% in 7 min	+1.5%
3.	M. K.	Cyst of left lung.	5900	197	191	35.1	94.4	44.1	87	87	0	+4% in 5 min	-4%
4.	V. Sh.	Cyst of left lung.	7300	250	234	34.2	94.1	35.8	108	100	8	+3.5% in 3 min	no change
5.	Ya. L.	Cancer of left lung (peripheral form).	6300	224	192	35.5	93.9	40.6	87	73	14	+5% in 3 min	-3%
6.	K. Sh.	Cancer of right lung (central form) with metastasis to mediastinum.	6230	246	172	39.4	88.1	42.3	85	66	19	+14% in 4 min	-2%
7.	F. B.	Cancer of right lung (peripheral form) with metastasis to mediastinum. Emphysema. Focal tuberculosis.	9160	312	307	34.0	83.6	42.0	85	69	16	+12% in 8 min	-4%
8.	Ya. Sh.	Cancer of right lung (central form) with metastasis to mediastinum and pleura.	9900	263	249	25.2	89.7	36.6	86	60	26	+10% in 2 min	+4%
	Ya. G.	Cancer of right lung.	5300	276	194	52.0	87.0	52.4	102	73	29	+8% in 2 min	-1%

We can see this even more clearly if we compare the cited data with the data in Table 3, where we have given the results of an examination of seven patients with lung cancer and two patients with a solitary cyst of the lungs. Among the latter virtually no deviations from the normal were observed. In the lung cancer patients, to judge from the pO_2 art. data, there were different degrees of hypoxemia, depending on the distribution of the cancer process as indicated in the literature [18 et al.]. The inspiration of 100% oxygen in almost all patients (except patient Ya. G.) gave practically total saturation, even with comparatively pronounced hypoxemia which excludes the possibility of a shunt. It should be pointed out that in these patients there was no clinical evidence of fresh inflammatory lesions in the lungs. Thus the idea developed that in inflammatory suppurative processes in the lungs it is the shunt which causes the hypoxemia to develop.

Examinations at different periods following surgery were made of 11 patients operated on for diseases of the lungs. The most pronounced lesions were observed in patients examined a month after surgery and showing hypoxemia (except patient G. Sh.) with relative hyperventilation and reduced respiratory efficiency. It should be pointed out that even in this case the breathing of oxygen did not give total saturation. In patients examined at later periods following surgery almost all the values for gas analysis returned to the normal or close to it (see Table 4).

TABLE 4

No.	Patient	Diagnosis	Min. volume ventilation in ml	Oxygen absorption in ml	Carbon dioxide secretion in ml	Oxyben utilization coefficient	Percentage of saturation in art. blood	pCO_2 alv.	pO_2 alv. effect.	pO_2 art.	AaD	Oxyhemometry: Breathing 100% Oxygen	Oxyhemometry: During Exercise
1.	G. Sh.	1 mo. after right lower lobectomy	7560	371	243	49.0	92.4	40.9	91	85	6	+4% in 5 min	+ 2%
2.	V. B.	1 mo. after left pneumonectomy	7950	200	—	25.1	94.5	42.0	86	76	10	+7% in 4 min	+ 1%
3.	E. L.	1 mo. after left upper lobectomy	7900	245	219	31.0	88.8	41.4	102	73	29	+5% in 2 min	- 1%
4.	E. K.	1 mo. after left pneumonectomy	6000	217	180	36.1	88.0	47.1	87	61	26	+3% in 7 min	- 1%
5.	E. G.	1 mo. after left lower lobectomy	9430	249	240	26.4	90.7	36.5	98	68	30	+8% in 8 min	—
6.	A. B.	1 yr. after left pneumonectomy	5460	350	—	64.0	90.8	37.2	83	72	11	+10% in 2 min	- 7%
7.	Ya. G.	1 yr. after right pneumonectomy	6700	193	192	28.8	90.2	37.4	80	76	4	+10% in 7 min	- 1%
8.	V. S.	6 mos. after left lower lobectomy	5900	239	233	38.8	92.5	40.7	86	80	6	+7.5% in 8 min	—
9.	S. S.	6 mos. after right upper lobectomy	6000	218	191	36.1	91.3	40.1	92	92	0	+9% in 10 min	- 1%
10.	V. K.	1 yr. after thoracotomy	6230	200	—	31.1	96.9	39.8	90	82	8	—	—
11.	L. S.	3 mos. after left upper lobectomy with thoracoplasty	8000	181	207	22.6	91.9	42.6	99	92	7	—	—

In conclusion we must point out that the small number of our observations does not allow us to draw definitive conclusions with relation to the mechanism by which hypoxemia arises; our aim was solely to employ a more accurate method in practice for obtaining data on the oxygen balance of the body.

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